



IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Application of: Judith A. Varner

Serial No.: 09/307,223

Filed: 05/07/99

Entitled:

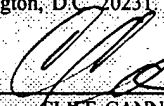
**METHODS FOR DETECTING AND
INHIBITING ANGIOGENESIS**

Group No.: 1642

Examiner: S. Ungar

**DECLARATION OF DR. VIRGIL L. WOODS, JR.
UNDER 37 C.F.R. §1.132**

Assistant Commissioner for Patents
Washington, D.C. 20231

CERTIFICATE OF MAILING UNDER 37 C.F.R. § 1.8(a)(1)(i)(A)	
I hereby certify that this correspondence (along with any referred to as being attached or enclosed) is, on the date shown below, being deposited with the U.S. Postal Service with sufficient postage as first class mail in an envelope addressed to: Assistant Commissioner for Patents, Washington, D.C. 20231.	
Dated: <u>February 19, 2002</u>	By:  CLIFF CANNON-CIN

Sir:

1. I, Dr. Virgil L. Woods, am the subject of the attached Curriculum Vitae and author of the publications shown on the list attached thereto. On the basis of the information and facts contained in these documents, I submit that I have been, and am, practicing in the field of integrin biology and biochemistry, and am qualified to speak on the level of ordinary skill in these fields.

2. I have read and understand the above-identified patent application, and pending claims as amended in the Amendment And Response which was mailed to the Office on September 5, 2001.

3. Claim 2 recites an agent which "does not substantially interfere with the specific binding of a ligand to an integrin other than $\alpha 5 \beta 1$ integrin to its ligand."

4. The Specification teaches that "As discussed for anti- $\alpha 5\beta 1$ antibodies, a peptide that specifically binds $\alpha 5\beta 1$ can be useful in a method of the invention where the antibody binds to $\alpha 5\beta 1$ with at least about a two-fold greater specificity than it binds to another integrin, . . . is more useful if it has at least about a five-fold greater specificity for $\alpha 5\beta 1$, and is particularly useful if it has at least about a one order of magnitude greater specificity for $\alpha 5\beta 1$ than for an integrin such as $\alpha V\beta 3$."¹

Based on this teaching, it is my understanding that Claim 2's term "**substantially**" refers to the exemplary preferred mathematical ranges (*i.e.*, at least about two-fold, five-fold, and ten-fold) for binding of the ligand (as exemplified by a peptide or antibody) to $\alpha 5\beta 1$ integrin, as compared to binding of the ligand to another integrin. From this teaching, it is my understanding that Claim 2's recitation of an agent which "does not substantially interfere with the specific binding of a ligand to an integrin other than $\alpha 5\beta 1$ integrin" means that the agent's interference with the specific binding of $\alpha 5\beta 1$ integrin to a ligand is at least two-fold greater than the interference of the agent with the specific binding of another integrin to its cognate ligand.

5. Claims 80-86, 90-96, and 110-116 recite that the binding of an "agent" to $\alpha 5\beta 1$ integrin is at least two-fold, five-fold, or ten-fold greater than binding of the agent to an integrin other than $\alpha 5\beta 1$ integrin.

6. The Specification teaches that "As disclosed herein, antibody, peptide and nonpeptide small organic molecule antagonists of $\alpha 5\beta 1$ can interfere with the specific binding of $\alpha 5\beta 1$ integrin with its ligands, particularly fibronectin, in vascular tissue, and can reduce or inhibit angiogenesis (see Examples II, III and IV). Such molecules that interfere with the specific binding of $\alpha 5\beta 1$ with its ligands are referred to herein generally as '*agents*.'"²

The Specification also teaches that "The term '*antagonist*' is used herein to mean an *agent*, which can be an antibody, a peptide or a nonpeptide small organic molecule."³

¹ Specification, page 23, lines 5-14.

² (Emphasis added) Specification, page 18, lines 7-15.

³ (Emphasis added) Specification, page 19, lines 24-27.

Based on these teachings, it is my understanding that the term "**agent**" of Claims 80-86, 90-96, and 110-116 is exemplified by an antibody, peptide, and/or nonpeptide small organic molecule.

7. The Specification teaches that "As discussed for anti- $\alpha 5\beta 1$ antibodies, a peptide that specifically binds $\alpha 5\beta 1$ can be *useful* in a method of the invention where the antibody binds to $\alpha 5\beta 1$ with at least about two-fold greater specificity than it binds to another integrin, . . . is more useful if it has at least about a five-fold greater specificity for $\alpha 5\beta 1$, and is particularly useful if it has at least about a one order of magnitude greater specificity for $\alpha 5\beta 1$ than for an integrin such as $\alpha V\beta 3$."⁴

Based on this teaching, it is my understanding that one property which makes an **antibody** or a **peptide** "useful" as an antagonist of $\alpha 5\beta 1$ binding to its ligand(s) is that the antibody or peptide binds with at least about two-fold greater, at least about five-fold greater, or at least about ten-fold greater specificity for $\alpha 5\beta 1$ than for $\alpha V\beta 3$. From this understanding in combination with my further understanding that an antibody and peptide are examples of an "agent," (as explained in item 6, *supra*), it is my opinion that the inventor contemplated that this desirable property (*i.e.*, relative binding to $\alpha 5\beta 1$ as compared to $\alpha V\beta 3$) which makes the exemplary antibodies and peptides "useful" in the claimed methods, is also a desirable property that is contemplated for **any other type of agent**, including the third exemplary type of nonpeptide small organic molecule. In other words, the Specification conveys to me that the inventor contemplated that one desirable property of **any agent** that interferes with the specific binding of $\alpha 5\beta 1$ integrin to its ligand is that the agent (regardless of its type) binds with at least about a two-fold greater, at least about five-fold greater, or at least about ten-fold greater specificity for $\alpha 5\beta 1$ than for $\alpha V\beta 3$.

8. Claims 86, 96, and 116 recite that an agent which interferes with specific binding of the $\alpha 5\beta 1$ integrin to a ligand "does not interfere with the specific binding of a ligand to an integrin other than $\alpha 5\beta 1$ integrin."

⁴ (Emphasis added) Specification, page 23, lines 5-14.

9. The Specification teaches that "Particularly useful antibodies for performing a method of the invention are those that specifically bind to an $\alpha 5\beta 1$ integrin. Such antibodies are particularly useful where they bind $\alpha 5\beta 1$ with at least an order of magnitude greater affinity than they bind another integrin, for example, $\alpha V\beta 3$ or $\alpha V\beta 5$. An anti-fibronectin antibody also can be *useful* in a method of the invention, particularly an anti-fibronectin antibody that interferes with *binding* of fibronectin to $\alpha 5\beta 1$ integrin, *but not to $\alpha V\beta 3$ or other integrin.*"⁵

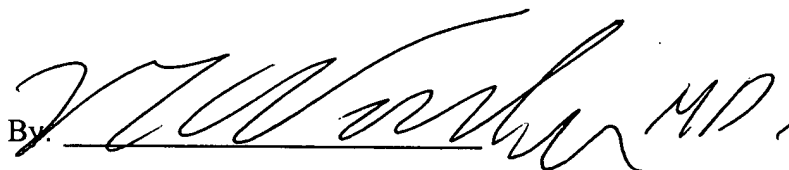
Based on this teaching, it is my understanding that one desirable property which makes an **antibody** "useful" in the claimed methods is that the antibody does not interfere with the specific binding of a ligand (such as fibronectin) to an integrin other than $\alpha 5\beta 1$. While this teaching refers to a desirable property of an antibody, it is nonetheless my opinion that this desirable property was contemplated by the inventor to apply to **any agent** because the Specification teaches that antibodies are an example of an agent (as explained *supra* in item 6).

10. I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under § 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the application or any patent issuing therefrom.

Dated: _____

1/2/2002

By: _____

 MD.

Dr. Virgil L. Woods

⁵ (Emphasis added) Specification, page 22, lines 15-19.

CURRICULUM VITAE

January 2, 2002

NAME: Virgil L. Woods, Jr., M.D.

SOCIAL SECURITY NO.: 563-70-6965

CURRENT POSITION: Associate Professor of Medicine & Orthopaedics
Division of Rheumatology
Department of Medicine
Department of Orthopaedics
University of California, San Diego

OFFICE ADDRESS: University of California, San Diego
Basic Science Building, Rm 5078
9500 Gilman Dr.
La Jolla, California 92093-0656

BUSINESS TELEPHONE: (619) 534-2180

FAX TELEPHONE: (619) 534-2606

EMAIL: vwoods@ucsd.edu

PERSONAL AND FAMILY:

BIRTH DATE: July 8, 1948

BIRTH PLACE: Oakland, California

MARITAL STATUS: Married, three children

EDUCATION:

1972 B.S., University of California, San Francisco, Major: Medical Sciences

1974 M.D., University of California, San Francisco, Major: Medicine

1979 B.A., University of California, San Diego, Major: Biochemistry

PROFESSIONAL EXPERIENCE:

1974 - 1975 Intern in Medicine, Barnes Hospital, St. Louis, Missouri

1975 - 1976 Resident in Medicine, Barnes Hospital, St. Louis, Missouri

1976 - 1979 Research Associate, Laboratory of Immunology, NIAID-NIH, Bethesda, MD

1979 - 1981 Rheumatology Fellow, University of California, San Diego

1981 - 1988 Assistant Professor of Medicine In Residence, University of California, San Diego

1988 - 1989 Associate Professor of Medicine in Residence, University of California, San Diego

1989 - 1992 Associate Professor of Medicine, University of California, San Diego

1992 - Present Associate Professor of Medicine and Orthopaedics, University of California, San Diego

MILITARY SERVICE:

July 1977 to U.S. Public Health Service
July 1979 Discharged with rank of Surgeon (Lt. Commander)

MEDICAL SPECIALTY BOARDS:

1978 American Board of Internal Medicine
1991 Subspecialty Boards in Rheumatology

FEDERAL GOVERNMENT PUBLIC ADVISORY COMMITTEES:
Grant Reviewer, Study Section for NIH NIAMD

MEMBERSHIP IN PROFESSIONAL SOCIETIES:

Member, American College of Rheumatology
Member, Alpha Omega Alpha Medical Society
Member, American Society of Hematology
Member, American Heart Association, Council on Thrombosis
Member, FASEB

REVIEWER FOR:

Arthritis and Rheumatism
Blood
Journal of Biological Chemistry
Protein Science
Experimental Cell Research

BIBLIOGRAPHY

ARTICLES

1. Woods, VL Jr, DE Nitecki and JW Goodman. The capacity of bifunctional antigens to bridge antibody molecules and to mediate cell cooperation. *Immunochem* 12:379-382, 1975.
2. Wilder, RL, CC Yuen, B Subbarao, VL Woods Jr, CB Alexander, and RC Mage. Tritium (3H) radiolabeling of protein-A and antibody to high specific activity: application to cell surface antigen radioimmunoassays. *J Immunol Meth* 28 (3-4):255-256, 1979.
3. Finkelman, FD, VL Woods Jr, A Berning, and I Scher. Demonstration of mouse serum IgD. *J Immunol* 123(3):1253-1259, 1979.
4. Kessler, SW, VL Woods Jr, FD Finkelman, and I Scher. Membrane orientation and location of multiple and distinct allotypic determinants of mouse lymphocyte IgD. *J Immunol* 123(6):2772-2778, 1979.
5. Finkelman, FD, JJ Mond, VL Woods Jr, S Wilburn, A Berning, E Sehgal, and I Scher. Effects of anti-immunoglobulin antibodies on murine B-lymphocyte and humoral immune responses. *Immunol Rev* 52:55, 1980.
6. Finkelman, FD, VL Woods Jr, S Wilburn, JJ Mond, KE Stein, A Berning, and I Scher. Augmentation of in vitro humoral immune responses in the mouse by an antibody to IgD. *J Exp Med* 152(3):493-506, 1980.
7. Woods, VL Jr, FD Finkelman, I Scher, and WE Paul. IgD allotypic determinants. I. Determinants expressed on murine IgD of the a allotype. *J Immunol* 125(6):2699-2707, 1980.
8. Woods, VL Jr, I Scher, and WE Paul. IgD allotypic determinants. II. Determinants expressed on IgD of mice of the b, e, and o Igh-C haplotypes. *J Immunol* 125(6):2708-2712, 1980.
9. Laskov, R, J Kim, VL Woods Jr, P McKeever, and R Asofsky. Spontaneous B lymphoma in aged BALB/c mice predominantly express membrane IgM and IgD with lambda type light chain. *Eur J Immunol* 11:462-468, 1981.
10. Bluestein, HG, and VL Woods Jr. Antineuronal antibodies in systemic lupus erythematosus. *Arthritis Rheum* 25:773-778, 1982.
11. Woods, VL Jr, EH Oh, D Mason, and R McMillan. Autoantibodies against the platelet glycoprotein IIb/IIIa complex in patients with chronic ITP. *Blood* 63:368-375, 1984.
12. Woods, VL Jr, Y Kurata, RR Montgomery, P Tani, D Mason, EH Oh, and R McMillan. Autoantibodies against platelet glycoprotein Ib in patients with chronic immune thrombocytopenic purpura. *Blood* 64:156-160, 1984.

13. Anderson, MJ, VL Woods Jr, P Tani, JM Lindstrom, D Schmidt, and R McMillan. Autoantibodies to platelet glycoprotein IIb/IIIa and to the acetylcholine receptor in a patient with chronic idiopathic thrombocytopenic purpura and myasthenia gravis. *Ann Intern Med* 100:829-831, 1984.
14. Woods, VL Jr, and R McMillan. Platelet autoantigens in chronic ITP (Annotation). *Brit J Haematol* 57:1-4, 1984.
15. Plow, EF, RP McEver, BS Collier, VL Woods Jr, GA Marguerie, and MH Ginsberg. Related binding mechanisms for fibrinogen, fibronectin, von Willebrand factor and thrombospondin on thrombin-stimulated human platelets. *Blood* 66:724-727, 1985.
16. Pischel, KD, HG Bluestein, and VL Woods Jr. Very late activation antigens (VLA) are human leukocyte-neuronal crossreactive cell surface antigens. *J Exp Med* 164:393-406, 1986.
17. Courtois, G, JJ Ryckewaert, VL Woods, MH Ginsberg, EF Plow, and GA Marguerie. Expression of intracellular fibrinogen on the surface of stimulated platelets. *Eur J Biochem* 159:61- 67, 1986.
18. Wencel-Drake, JD, EF Plow, TJ Kunicki, VL Woods Jr, DM Keller, and MH Ginsberg. Localization of internal pools of membrane glycoproteins involved in platelet adhesive responses. *Amer J Pathol* 124:324-334, 1986.
19. Bluestein, HG, KD Pischel, and VL Woods Jr. Immunopathogenesis of the neuropsychiatric manifestations of systemic lupus erythematosus. *Springer Semin Immunopathol* 9:237-249, 1986.
20. Woods, VL Jr, LE Wolff, and DM Keller. Resting platelets contain a substantial centrally located pool of glycoprotein IIb-IIIa complex which may be accessible to some but not other extracellular proteins. *J Biol Chem* 261:15242-15251, 1986.
21. Pischel, KD, ME Hemler, C Huang, HG Bluestein, and VL Woods Jr. Use of the monoclonal antibody 12F1 to characterize the differentiation antigen VLA-2. *J Immunol* 138:226-233, 1987.
22. Pischel, KD, SD Marlin, TA Springer, VL Woods Jr, and HG Bluestein. Polymorphism of lymphocyte function associated antigen-1, LFA-1 demonstrated by a lupus patient's alloantiserum. *J Clin Invest* 79:1607-1614, 1987.
23. Tsubakio, T, P Tani, VL Woods Jr, and R. McMillan. Autoantibodies against platelet GPIIb/IIIa in chronic ITP react with different epitopes. *Br J Haematol* 67:345, 1987.
24. McMillan, R, P Tani, F Millard, P Berchtold, L Renshaw, and VL Woods Jr. Platelet-associated and plasma anti-glycoprotein autoantibodies in chronic ITP. *Blood* 70:1040, 1987.
25. Pischel, KD, HG Bluestein, and VL Woods Jr. Platelet glycoproteins Ia, Ic, and IIa are physicochemically indistinguishable from the very late activation antigens adhesion-related proteins of lymphocytes and other cell types. *J Clin Invest* 81:505-513, 1988.
26. Santoro, SA, SM Rajpara, WD Staats, and VL Woods, Jr. Isolation and characterization of a

platelet surface collagen binding complex related to VLA-2. *Biochem Biophys Res Comm* 153:217-228, 1988.

27. Woods, VL Jr., KD Pischel, ED Avery, and HG Bluestein. Antigenic polymorphism of human very late activation protein-2 (platelet glycoprotein Ia-IIa). Platelet alloantigen Hca. *J Clin Invest* 83:979-985, 1989.
28. Savage, B, C Hunter, LA Harker, VL Woods, Jr. and SR Hanson. Thrombin-induced increase in surface expression of epitopes on platelet membrane glycoprotein IIb/IIIa complex and GMP-140 is a function of platelet age. *Blood*, 74(3):1007-1014, 1989.
29. Smith, JW, JG Kelton, P Horsewood, C Brown, A Giles, D Hunter, R Meyer, V Woods, and R Burrows. Platelet specific alloantigens (Zava/Zavb) on the platelet glycoprotein Ia/IIa complex. *Br J Haematol* 72:4, 1989.
30. Schorer, AE, PG Duane, VL Woods, DE Niewoehner. Antiphospholipid antibodies inhibit phospholipase A2 activity. *J Lab Clin Med* 120:67-77, 1992.
31. Gesink, DS, HO Pacheco, S Kuiper, D Amiel, WH Akeson, and VL Woods, Jr. Immunohistochemical localization of $\beta 1$ integrins in anterior cruciate and medial collateral ligaments of the human and rabbit. *J Ortho Res* 10:596-599, 1992.
32. Berlin, OB, D Samid, R Donthineni-Rao, WH Akeson, D Amiel, VL Woods Jr. Development of a novel spontaneous metastasis model of human osteosarcoma transplanted orthotopically into bone of nude mice. *Cancer Research* 53:4890-4895, 1993.
33. Woods, VL, Jr, PJ Schreck, DS Gesink, HO Pacheco, D Amiel, WH Akeson, and M Lotz. Integrin expression by human articular chondrocytes. *Arthritis Rheum* 37:537-544, 1994.
34. Chen, PP, DA Carson, P Fischer, HH Handley, SJC Leu, R Mcmillan, W Novotny, T Olee, and VL Woods. Molecular genetics or human autoantibodies. *Pathol Biol*, 42(8):798-800, 1994.
35. Schreck, PJ, LR Kitabayashi, D Amiel, WH Akeson, and VL Woods Jr. Integrin receptor display increases in the wounded rabbit medial collateral ligament but not in the anterior cruciate ligament. *J Orthop Res*, 13:174-183, 1995.
36. Ruoslahti, E, M Pierschbacher, and VL Woods Jr. The $\alpha 5 \beta 1$ integrin in tumour suppression. *Bull. Inst Pasteur*, 92(4):242-247, 1995.
37. Maemura, M, SK Akiyama, VL Woods, Jr, RB Dickson. Expression and ligand binding of $\alpha 2 \beta 1$ integrin on breast carcinoma cells. *Clin Exp Metastasis*, 13:223-235, 1995.
38. AbiEzzi, SS, DS Gesink, PJ Schreck, D Amiel, WH Akeson, and VL Woods Jr. Increased expression of the $\beta 1$, $\alpha 5$, and αv integrin adhesion receptor subunits occur coincident with remodeling of stress-deprived rabbit anterior cruciate and medial collateral ligaments. *J Orthop Res*, 13:594-601, 1995.

39. Woolf, NK, DV Jaquish, FJ Koehn, VL Woods, and DA Peterson. Improved resolution of fibronectin mRNA expression in the inner ear using laser scanning confocal microscopy. *J Histochem Cytochem*, 44(1):27-34, 1996.
40. Rennekampff HO, JF Hansbrough, V Kiessig, S Abiezzi, and V Woods, Jr. Wound closure with human keratinocytes cultured on a polyurethane dressing overlaid on a cultured human dermal replacement. *Surgery*, 120:16-22, 1996.
41. Olee, T, SS Pierangeli, HH Handley, DT Le, X Wei, CJ Lai, J En, W Novotny, EN Harris, VL Woods, Jr., and P Chen. A monoclonal IgG anticardiolipin antibody from a patient with the antiphospholipid syndrome is thrombogenic in mice. *Proc Natl Acad Sci USA*, 93:8606-8611, 1996.
42. Pasqualini R, S Bourdoulous, E Koivunen, VL Woods, Jr, and E Ruoslahti. A polymeric form of fibronectin has anti-metastatic effects against multiple tumor types. *Nature Medicine*, 2(11):1197-1203, 1996.
43. Mo L, SC Leu, C Berry, F Liu, T Olee, Y Yang, DS Beardsley, R Mcmillan, VL Woods Jr, PP Chen. The frequency of homozygous deletion of a developmentally regulated Vh Gene (Humh3005) is increased in patients with chronic idiopathic thrombocytopenic purpura. *Autoimmunity*, 24:257-263, 1996.
44. Olee T, J En, CJ Lai, L Mo, CS Cho, X Wei, XF Wang, VL Woods, and PJ Chen. Generation and analysis of an IgG anti-platelet autoantibody reveals unusual molecular features. *Br J Haematol*, 96(4):836-845, 1997.
45. Rennekampff, HO, JF Hansbrough, V Woods, C Dore, V Kressig and JM Schroder. Role of melanoma growth stimulatory activity (MGSA/gro) on keratinocyte function in wound healing. *Arch Dermatol Res* 289:204, 1997.
46. Yang, Y-Y, P Fischer, S-J Leu, M Zhu, VL Woods, Jr., and PP Chen. Possible presence of enhancing antibodies in idiopathic thrombocytopenic purpura. *Br J Haematol*, 104:69-80, 1999.
47. Zhu, M, T Olee, DT Le, RAS Roubey, BH Hahn, VL Woods, and PP Chen. Characterization of IgG monoclonal anti-cardiolipin/anti-beta(2)GP1 antibodies from two patients with antiphospholipid syndrome reveals three species of antibodies. *Br J Haematol*, 105(1):102-109, 1999.
48. Rosfjord, EC, M Maemura, MD Johnson, JA Torri, SK Akiyama, VL Woods, and RB Dickson. Activation of protein kinase C by phorbol esters modulates alpha(2)beta(1) integrin on MCF-7 breast cancer cells. *Exp Cell Res*, 248(1):260-271, 1999.
49. Mills, PJ, AS Maisel, MG Ziegler, JE Dimsdale, S Carter, B Kennedy, VL Woods, Jr. Peripheral blood mononuclear cell-endothelial adhesion in human hypertension following exercise. *J Hypertension*, 18:1801-1806, 2000.
50. Horkko, S, T Olee, L Mo, DW Branch, VL Woods, Jr, W Palinski, PP Chen, JL Witztum. Anticardiolipin antibodies from patients with the antiphospholipid antibody syndrome recognize epitopes in both β_2 -glycoprotein 1 and oxidized low-density lipoprotein. *Circulation*, 104:941-946, 2001.

PATENTS

1. Woods, VL. Method for characterization of the fine structure of protein binding sites. U.S. Patent # 5,658,739, filed May 10, 1994, issued August 19, 1997 : Assignee: The Regents of the University of California, 1997.
2. Woods, VL. Methods for the high- resolution identification of solvent- accessible amide hydrogens in polypeptides or proteins and for the characterization of the fine structure of protein binding sites. U.S. Patent # 6,291,189, issued Sept. 18, 2001
3. Woods, VL. Method for characterization of the fine structure of protein binding sites. U.S. Patent # 6,331,400, filed July 16, 1997, issued December 18, 2001 : Assignee: Carta Proteomics, Inc.

BOOKS AND CHAPTERS

1. Zvaifler, NJ, and VL Woods Jr. Etiology and pathogenesis of systemic lupus erythematosus. In: Textbook of Rheumatology, chap. 68, second edition. WN Kelley, ED Harris, Jr., S Ruddy, and CB Sledge, eds. W.B. Saunders Co., Philadelphia, pp. 1042-1070, 1985.
2. Woods, VL Jr., NJ Zvaifler. Etiology and pathogenesis of systemic lupus erythematosus. In: Textbook of Rheumatology, chap. 63, third edition. WN Kelley, ED Harris, Jr., S Ruddy, and CB Sledge, eds. W.B. Saunders Co., Philadelphia, pp. 1077-1100, 1989.
3. Woods, VL Jr. Pathogenesis of systemic lupus erythematosus. In: Textbook of Rheumatology, Chapter 60, 4th Edition. WN Kelley, ED Harris, Jr., S Ruddy, and CB Sledge, eds. W.B. Saunders Co., Philadelphia, pp. 999-1016, 1993.
4. Schreck, PJ, D Amiel, VL Woods Jr, and WH Akeson. The role of integrin adhesion receptors in anterior cruciate ligament physiology. In: The Anterior Cruciate Ligament: Current and Future Concepts. Section II, Subsection C, Chapter 38. D Jackson (ed), Raven Press, pp. 423-430, 1993.
5. Woods, VL Jr., P Schreck, D Amiel, WH Akeson, and M Lotz. The role of integrin adhesion receptors in aging of cartilage and osteoarthritis. In: Musculoskeletal Soft-Tissue Aging: Impact on Mobility. JA Buckwalter, VM Goldberg, SL-Y Woo, eds. American Academy of Orthopaedic Surgeons, Rosemont, Illinois, pp. 165-182, 1993.
6. Woods, VL Jr. Human systemic lupus erythematosus. In: The Molecular Pathology of Autoimmune Diseases, Chapter 19. CA Bona, KA Siminovitch, M Zanetti, AN Theofilopoulos, eds. Harwood Academic Publishers, Chur, Switzerland, pp. 265-280, 1993.
7. Berlin, O, B Gunterberg, D Samid, VL Woods, and WH Akeson. Effect of suramin in an orthotopic model of human osteosarcoma growing in the tibia of athymic mice. In: Limb Savage, Current Trends, Proceedings of the 7th International Symposium, Singapore, pp. 33-37,

August 23-27, 1993.

ABSTRACTS AND REVIEW ARTICLES

1. Finkelman, FD, VL Woods Jr, A Berning, and I Scher. Mice have serum IgD. *J Supramol Struct*, abstract #635, p. 250, 1979.
2. Woods, VL Jr, FD Finkelman, A Berning, I Scher, and WE Paul. Mouse serum IgD: Assay and ontogeny. *Fed Proc* 38:1464, 1979.
3. Scher, I, JJ Mond, VL Woods Jr, S Wilburn, and FD Finkelman. Augmentation of murine in vivo antibody responses after anti-delta modulation of B-cell surface IgD. *Fed Proc* 39:806, 1980.
4. Scher, I, JJ Mond, AK Berning, VL Woods Jr, K Stein, S Wilburn, and FD Finkelman. Effect of anti-IgD antibody on in vivo murine humoral immune responses. In: Abstracts - 4th International Congress of Immunology. JL Preud'homme and VAL Hawken, eds., 1980.
5. Woods, VL Jr, and HG Bluestein. Characterization of neuronal and lymphoid membrane molecules reactive with antisera to SLE cryoglobulins. *Arthritis Rheum* 24:S81, 1981.
6. Woods, VL Jr, EH Oh, D Mason, and R McMillan. Autoantibodies against the platelet glycoprotein IIb/IIIa complex (GPIIb/GPIIIa) in patients with chronic idiopathic thrombocytopenic purpura (ITP). *Blood* 60:192a, 1982.
7. Woods, VL Jr, MH Ginsberg, TJ Kunicki, EH Oh, and BC Jaques. Demonstration of hybridoma antibodies reactive only with the complexed state of platelet glycoproteins IIb and IIIa. *Blood* 60:205a, 1982.
8. Pischel, KD, HG Bluestein, and VL Woods Jr. Molecular identification of lymphocyte cell surface molecules reactive with systemic lupus erythematosus (SLE) autoantibodies. *Clin Res* 32:(1)41A, 1984.
9. Pischel, KD, HG Bluestein, and VL Woods Jr. Characterization of lymphocyte cell surface proteins reactive with systemic lupus erythematosus (SLE) antibodies. *Clin Res* 32:538A, 1984.
10. Pischel, KD, HG Bluestein, and VL Woods Jr. Molecular characterization of lymphocyte cell surface proteins reactive with systemic lupus erythematosus (SLE) autoantibodies. *Arthritis Rheum* 27:S83, 1984.
11. Tsubakio, T, R McMillan, P Tani, and VL Woods. Platelet antigens in chronic ITP. *Clin Res* 32:325A, 1984.
12. Woods, VL Jr, Y Kurata, T Tsubakio, RR Montgomery, P Tani, and R McMillan. Autoantibodies against platelet glycoproteins in chronic immune thrombocytopenic purpura (C-ITP). *Blood* 62 (suppl. 1):249a, 1984.
13. Wencel-Drake, JD, T Kunicki, VL Woods Jr, and MH Ginsberg. Evidence for distinct

intracellular pools of platelet membrane glycoproteins GPIb and GPIIb/III. *Blood* 62 (suppl 1):270a, 1984.

14. Wencel-Drake, JD, T Kunicki, V Woods, EF Plow, and MH Ginsberg. Intracellular pools of platelet membrane glycoproteins GPIb and GPIIb/III. *Fed Proc* 43(3):660, 1984.
15. Keller, DM, MH Ginsberg, EF Plow, and VL Woods Jr. Thrombin can induce cell surface display of a pool of platelet glycoprotein IIb and IIIa heterodimer distinct from that on the surface of resting platelets. *Blood* 64:248a, 1984.
16. Woods, VL Jr. Platelet GPIIb/GPIIIa-specific monoclonal antibodies can gain access to a substantial internal pool of GPIIb/GPIIIa when platelets are in the intact resting state. *Thrombo Haemo* 54(1):112, 1985.
17. Pischel, KD, HG Bluestein, and VL Woods Jr. Lymphocytes bear molecules (VLA) that are antigenically and structurally similar to platelet GPIa, GPIc-alpha, and GPIIa. *Blood* 66:311a, 1985.
18. Pischel, KD, HG Bluestein and VL Woods Jr. VLA-like membrane proteins on lymphocyte and neuronal cells react with systemic lupus erythematosus antibodies. *Clin Res* 33:(2)592A, 1985.
19. Pischel, KD, HG Bluestein, and VL Woods Jr. Systemic lupus erythematosus (SLE) antibody reactivity with VLA-like membrane proteins on lymphocyte and neuronal cells. *Arthritis Rheum* 28:S92, 1985.
20. Pischel, KD, HG Bluestein, and VL Woods Jr. A patient with SLE has serum antibodies reactive with an LFA-1-like 175/95 kd glycoprotein. *Arthritis Rheum* 29:S23, 1986.
21. Pischel, KD, HG Bluestein, VL Woods Jr, and TA Springer. Human LFA-1 variation revealed by antibodies from a patient with systemic lupus erythematosus. *Clin Res* 34:503A, 1986.
22. Pischel, KD, HG Bluestein, VL Woods Jr, and TA Springer. Heterogeneity of human LFA-1 glycoproteins revealed by antibodies in an SLE serum. *Fed Proc* 45:992, 1986.
23. McMillan, R, P Tani, L Renshaw, and VL Woods. Platelet-associated and plasma anti-glycoprotein autoantibodies in chronic ITP. *Blood* 68:113A, 1986.
24. Woods, VL Jr, KD Pischel, and HG Bluestein. Polymorphism of human VLA antigens revealed by alloantibodies from systemic lupus erythematosus sera. *Clin Res* 35:436A, 1987.
25. Pischel, KD, VL Woods Jr, and HG Bluestein. Human VLA antigen polymorphisms revealed by systemic lupus erythematosus sera alloantibodies. *Arthritis Rheum* 30:S47, June 9, 1987.
26. Brodie, CJ, VL Woods, and HG Bluestein. Characterization of neuronal antigens reactive with antibodies in SLE serum and CSF. *Arthritis Rheum* 31:S107, 1988.
27. Mizutani, WT, VL Woods, JA McCutchan, and NJ Zvaifler. Anticardiolipin antibodies in human immunodeficiency virus (HIV) infected gay males may be associated with a deteriorating clinical course. *Arthritis Rheum* 31:S35, 1988.

28. Mizutani, WM, VL Woods, and NJ Zvaifler. Anticardiolipin antibodies in human immunodeficiency virus (HIV) infected gay males may be associated with a deteriorating clinical course. Clin Res 36:188a, 1989.
29. Jarjour, WN, V Tsai, VL Woods, Jr., W Welch, S Pierce, M Shaw, H Mehta, W Dillman, NJ Zvaifler and J Winfield. Cell surface expression of heat shock proteins. Arthritis Rheum 32:544, 1989.
30. Gesink, DS, HO Pacheco, VL Woods, D Amiel, WH Akeson. Immuno-histochemical localization of VLA integrins in anterior cruciate new and medial collateral ligaments of human and rabbit. Combined meeting of the Orthopedic Research Societies of USA, Japan, and Canada in Bannf, Alberta, p. 11, Oct.21-23, 1991.
31. Pacheco, HO, DS Gesink, VL Woods, D Amiel, WH Akeson. Integrin expression in normal human articular chondrocytes. Combined meeting of the Orthopedic Research Societies of USA, Japan, and Canada in Banff, Alberta, p. 3, Oct. 21-23, 1991.
32. Pacheco, HO, DS Gesink, VL Woods, CN Nagineni, D Amiel, WH Akeson. Integrin and fibronectin isoform expression in cultured rabbit anterior cruciate and medial collateral ligament fibroblasts. Combined meeting of the Orthopedic Research Societies of USA, Japan, and Canada in Banff, Alberta, p. 102, Oct. 21-23, 1991.
33. Woods, VL Jr, DS Gesink, HO Pacheco, D Amiel, WH Akeson, and M Lotz. Integrin expression by human articular chondrocytes. Arthritis Rheum 34:S40, 1991.
34. Schreck, PJ, VL Woods, Jr, DS Gesink, HO Pacheco, D Amiel, M Lotz, and WH Akeson. Integrin expression by human articular chondrocytes. West Connect Tiss Soc Mtng, San Diego, CA May 22-24, 1992.
35. Schreck, PJ, DS Gesink, HO Pacheco, VL Woods, Jr, and D Amiel. $\beta 1$ integrin expression in the anterior cruciate and medial collateral ligaments of human and rabbit. West Connect Tiss Soc Mtng, San Diego, CA, May 22-24, 1992.
36. AbiEzzi, SS, DS Gesink, PJ Schreck, D Amiel, WH Akeson, and VL Woods Jr. Integrin expression in the anterior cruciate (ACL) and medial collateral (MCL) ligaments after joint immobilization. 40th Annual Mtng Orthop Res Soc, February 21-24, New Orleans, Orthop Trans, 19(2):299, 1994.
37. Gesink, DS, LR Kitabayashi, JB Massie, D Amiel, VL Woods Jr., and WH Akeson. Integrin expression in injured intervertebral discs of rabbit. 40th Annual Mtng Orthop Res Soc, February 21-24, New Orleans, 19(1):90, 1994.
38. Gesink, DS, LR Kitabayashi, D Amiel, VL Woods Jr., and WH Akeson. Integrin expression in human meniscus. 40th Annual Mtng Orthop Res Soc, February 21-24, New Orleans, 19(2):300, 1994.
39. AbiEzzi, SS, DS Gesink, PJ Schreck, D Amiel, WH Akeson, and VL Woods Jr. Integrin expression in the stress-deprived anterior cruciate (ACL) and medial collateral (MCL) ligaments. FASEB J A102, March 1994.

40. AbiEzzi, SS, DS Gesink, PJ Schreck, D Amiel, WH Akeson, and VL Woods Jr. Increased integrin adhesion receptor expression occurs coincident with remodelling of stress-deprived rabbit anterior cruciate (ACL) and medial collateral (MCL) ligaments. SIROT Meeting, Boston, October 26-29, 1994.
41. Amiel, D, SS AbiEzzi, WH Akeson, and VL Woods Jr. The role of integrin adhesion receptors expression in periarticular ligaments during stress deprivation. FECTS Meeting, Lyon, France, August 30-September 3, 1994.
42. Woods, VL Jr, M Lotz, and SS AbiEzzi. Ageing of human cartilage is accompanied by a selective decrease in chondrocyte expression of α_v -subunit containing integrin adhesion receptors. J Invest Med, 43(S2):308A, 1995.
43. AbiEzzi, SS, D. Amiel, WH Akeson, and VL Woods Jr. Ageing of human articular cartilage is accompanied by a selective decrease in chondrocyte expression of α_v -containing integrin adhesion receptors. 2nd Combined Meeting of the Orthopedic Research Societies of USA, Japan, Canada, and Europe, November 5-8, 1995.
44. Woods, Jr. VL, M Lotz and SS AbiEzzi. Chondrocyte expression of α_v subunit-containing integrins selectively decreases with ageing. Arth Rheum (S9)159, 1995.
45. AbiEzzi, SS, D Amiel, WH Akeson, and VL Woods Jr. Both anterior cruciate and medial collateral ligament fibroblasts can upregulate their integrin adhesion receptors when properly stimulated. Orthopedic Research Society Meeting, Orlando, FL. Orthop. Trans. 19(4):889, 1996.
46. Yang, YY, S-JC Leu, P Fischer, VL Woods Jr, PP Chen. Molecular analysis of phage library-derived IgG rheumatoid factors isolated from an idiopathic thrombocytopenic purpura patient. FASEB 12(4):A608, 1998.